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☐ 1. Document ID: US 20020182673 A1

L3: Entry 1 of 30 File: PGPB Dec 5, 2002

PGPUB-DOCUMENT-NUMBER: 20020182673
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020182673 A1

TITLE: IL-17 homologous polypedies and therapeutic uses thereof

PUBLICATION-DATE: December 5, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Chen, Jian	Princeton	NJ	US	
Filvaroff, Ellen	San Francisco	CA	US	
Fong, Sherman	Alameda	CA	US	
French, Dorothy	Redwood City	CA	US	
Goddard, Audrey	San Francisco	CA	US	
Godowski, Paul J.	Hillsborough	CA	US	
Grimaldi, J. Christopher	San Francisco	CA	US	
Gurney, Austin L.	Belmont	CA	US	
Hillan, Kenneth J.	San Francisco	CA	US	
Hymowitz, Sarah G.	San Francisco	CA	US	
Li, Hanzhong	San Mateo	CA	US	
Pan, James	Zitobicoke	CA	CA	
Starovasnik, Melissa A.	San Francisco	CA	US	
Tumas, Daniel	Orinda	CA	US	
Van Lookeren, Menno	San Francisco	CA	US	
Vandlen, Richard	Hillsborough	CA	US	
Watanabe, Colin K.	Moraga	CA	US	
Williams, P. Mickey	Half Moon Bay	CA	US	
Wood, William I.	Hillsborough	CA	US	
Yansura, Daniel G.	Pacifica		US	

US-CL-CURRENT: 435/69.1; 435/320.1, 435/325, 530/350, 536/23.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | KWIC | Draw Desc | Image

☐ 2. Document ID: US 20020177551 A1

L3: Entry 2 of 30 File: PGPB Nov 28, 2002

PGPUB-DOCUMENT-NUMBER: 20020177551
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020177551 A1

WEST Search History

DATE: Thursday, December 05, 2002

Set Name side by side	Query	Hit Count	Set Name result set
DB=USPT,PGPB,EPAB,DWPI,TDBD; THES=ASSIGNEE; PLUR=YES; OP=ADJ			
L5	L4 and (CD40 adj binding same protein?)	14	L5
L4	CD40 same disease?	160	L4
L3	L2 and disease?	30	L3
L2	TNF same (superfamily or family) and binding?	52	L2
L1	CD40 adj binding adj protein?	9	L1

END OF SEARCH HISTORY

Inventor Name Search Result

Your Search was:

Last Name = PYPE
First Name = STEFAN

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09697863	Not Issued	071	10/27/2000	CD40-INTERACTING AND TRAF-INTERACTING PROTEINS	PYPE, STEFAN M C

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** pype **First Name** stefan Search

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Inventor Name Search Result

Your Search was:

Last Name = REMACLE
First Name = JACQUES

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09449285	6313280	150	11/24/1999	SMAD-INTERACTING POLYPEPTIDES AND THEIR USE	REMACLE , JACQUES
09964238	Not Issued	030	09/26/2001	SMAD-INTERACTING POLYPEPTIDES AND THEIR USE	REMACLE, JACQUES
10028396	Not Issued	030	12/21/2001	NUCLEIC ACID BINDING OF MULTI-ZINC FINGER TRANSCRIPTION FACTORS	REMACLE, JACQUES
09697863	Not Issued	071	10/27/2000	CD40-INTERACTING AND TRAF-INTERACTING PROTEINS	REMACLE, JACQUES E F

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** remacle **First Name** jacques Search

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Inventor Name Search Result

Your Search was:

Last Name = HUYLEBROECK
First Name = DANNY

Application#	Patent#	Status	Date Filed	Title	Inventor Name
09449285	6313280	150	11/24/1999	SMAD-INTERACTING POLYPEPTIDES AND THEIR USE	HUYLEBROECK , DANNY
09964238	Not Issued	030	09/26/2001	SMAD-INTERACTING POLYPEPTIDES AND THEIR USE	HUYLEBROECK, DANNY
10028396	Not Issued	030	12/21/2001	NUCLEIC ACID BINDING OF MULTI-ZINC FINGER TRANSCRIPTION FACTORS	HUYLEBROECK, DANNY
09697863	Not Issued	071	10/27/2000	CD40-INTERACTING AND TRAF-INTERACTING PROTEINS	HUYLEBROECK, DANNY F E

Inventor Search Completed: No Records to Display.

Search Another: Inventor **Last Name** huylebroeck **First Name** danny Search

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FILE 'HOME' ENTERED AT 20:55:56 ON 05 DEC 2002

=> index bioscience medicine pharmacology meetings
FILE 'DRUGMONOG' ACCESS NOT AUTHORIZED
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.21       0.21

INDEX 'ADISALERTS, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI,
       BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA,
       CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DDFB,
       DDFU, DGENE, DRUGB, DRUGLAUNCH, DRUGMONOG2, ...'
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83 FILES IN THE FILE LIST IN STNINDEX

Enter SET DETAIL ON to see search term postings or to view
search error messages that display as 0* with SET DETAIL OFF.

=> s (CD40 (w) binding (w) protein?) and (tumor (w)  necrosis (w)  factor (s)( family  or
superfamily))
          1   FILE AGRICOLA
          3   FILE BIOSIS
          3   FILE BIOTECHNO
  12 FILES SEARCHED...
          1   FILE CANCERLIT
          3   FILE CAPLUS
  17 FILES SEARCHED...
  24 FILES SEARCHED...
          3   FILE EMBASE
          3   FILE ESBIOBASE
  33 FILES SEARCHED...
          0*  FILE FEDRIP
          3   FILE LIFESCI
  44 FILES SEARCHED...
          5   FILE MEDLINE
  50 FILES SEARCHED...
          3   FILE SCISEARCH
          2   FILE TOXCENTER
          5   FILE USPATFULL
  60 FILES SEARCHED...
          2   FILE WPIDS
          2   FILE WPINDEX
  71 FILES SEARCHED...

  14 FILES HAVE ONE OR MORE ANSWERS,   83 FILES SEARCHED IN STNINDEX

L1    QUE (CD40 (W) BINDING (W) PROTEIN?) AND (TUMOR (W) NECROSIS (W) FACTOR (S)
          (FAMILY OR SUPERFAMILY))

=> file hits
COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         9.54       9.75

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FILE 'WPINDEX' ACCESS NOT AUTHORIZED

FILE 'AGRICOLA' ENTERED AT 21:07:19 ON 05 DEC 2002

FILE 'CANCERLIT' ENTERED AT 21:07:19 ON 05 DEC 2002

=> s l1
L2 5 FILE MEDLINE
L3 5 FILE USPATFULL
L4 3 FILE BIOSIS
L5 3 FILE BIOTECHNO
L6 3 FILE CAPLUS
L7 3 FILE EMBASE
L8 3 FILE ESBIOBASE
L9 3 FILE LIFESCI
L10 3 FILE SCISEARCH
L11 2 FILE TOXCENTER
L12 2 FILE WPIDS
L13 1 FILE AGRICOLA
L14 1 FILE CANCERLIT

TOTAL FOR ALL FILES
L15 37 L1

=> dup rem l15
PROCESSING COMPLETED FOR L15
L16 12 DUP REM L15 (25 DUPLICATES REMOVED)

=> d l16 1-12 ibib abs

L16 ANSWER 1 OF 12 USPATFULL
ACCESSION NUMBER: 2002:78211 USPATFULL
TITLE: Method for treatment of tumors using photodynamic therapy
INVENTOR(S): Fanslow, William C., III, Normandy Park, WA, UNITED STATES
Thomas, Elaine K., Seattle, WA, UNITED STATES
PATENT ASSIGNEE(S): IMMUNEX CORPORATION, Seattle, WA, UNITED STATES (U.S. corporation)

NUMBER	KIND	DATE

PATENT INFORMATION: US 2002041864 A1 20020411
APPLICATION INFO.: US 2001-842745 A1 20010425 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 2000-199545P	20000425 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: APPLICATION
LEGAL REPRESENTATIVE: IMMUNEX CORPORATION, LAW DEPARTMENT, 51 UNIVERSITY STREET, SEATTLE, WA, 98101
NUMBER OF CLAIMS: 22
EXEMPLARY CLAIM: 1
LINE COUNT: 889
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB A method for treating tumor-bearing subjects that includes administering to the tumor bearing subject a therapeutically effective amount of a **CD40 binding protein** in conjunction with photodynamic therapy, is disclosed.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 2 OF 12 USPATFULL
ACCESSION NUMBER: 2002:303718 USPATFULL
TITLE: Methods of reducing bone loss with CD40 ligand
INVENTOR(S): Ahuja, Seema A., San Antonio, TX, United States
Bonewald, Lynda F., San Antonio, TX, United States
PATENT ASSIGNEE(S): Board of Regents, The University of Texas System, Austin, TX, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6482411	B1	20021119
APPLICATION INFO.:	US 2000-645926		20000824 (9)

	NUMBER	DATE
PRIORITY INFORMATION:	US 1999-151250P	19990827 (60)

DOCUMENT TYPE: Utility
FILE SEGMENT: GRANTED
PRIMARY EXAMINER: Gambel, Phillip
LEGAL REPRESENTATIVE: Williams, Morgan and Amerson
NUMBER OF CLAIMS: 34
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 5120
AB Provided are methods and compositions using one or more CD40 agonists, such as CD40 ligands and/or agonistic anti-CD40 antibodies, to reduce or prevent cell death, or apoptosis, in bone cells. Methods of treating or preventing bone loss, including osteoporosis, as well as methods of reducing or eliminating the bone loss associated with steroid administration are particularly provided. Further disclosed are a variety of therapeutic kits and cocktails.

L16 ANSWER 3 OF 12 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2001-235057 [24] WPIDS
DOC. NO. CPI: C2001-070422
TITLE: Modulating the immune system, useful e.g. for treating cancer or infections, by administering an antigen and agent that binds or activates CD40, inducing cytotoxic T cells.
DERWENT CLASS: B04 D16
INVENTOR(S): OHASHI, P S
PATENT ASSIGNEE(S): (UYHE-N) UNIV HEALTH NETWORK
COUNTRY COUNT: 90
PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 2001015728	A1	20010308	(200124)*	EN	67

RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW MZ NL OA PT SD SE SL SZ TZ UG ZW

W: AE AL AM AT AU AZ BA BB BG BR BY CA CH CN CR CU CZ DE DK DM EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT TZ UA UG UZ VN YU ZA ZW

AU 2000066770 A 20010326 (200137)

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 2001015728	A1	WO 2000-CA960	20000823
AU 2000066770	A	AU 2000-66770	20000823

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 2000066770	A	Based on	WO 200115728

PRIORITY APPLN. INFO: US 1999-384862 19990827

AN 2001-235057 [24] WPIDS

AB WO 200115728 A UPAB: 20010502

NOVELTY - Modulating the immune system by administering an antigenic molecule (I) to generate antigen-presenting cells (APC) that present (part of) (I) on their surface and administering a CD40-binding or -activating molecule (II), specific for APC that display (I) in vivo, is used to prime activation of a cytotoxic T lymphocyte (CTL)-mediated, antigen-specific immune response.

DETAILED DESCRIPTION - An INDEPENDENT CLAIM is also included for increasing the in vivo effect of a vaccine (A) by administering it in conjunction with (II).

ACTIVITY - Immunomodulatory; anticancer; antibacterial; antiviral; antiparasitic; antifungal.

MECHANISM OF ACTION - Activation of CTL to target and destroy antigen-expressing cells. Treatment with (II) increases interferon production and prevents induction of tolerance.

Transgenic Bln/TCR mice (Cell, 65 (1991) 305) expressed the p33 glycoprotein of lymphocytic choriomeningitis virus in the pancreas, under control of the rat insulin II promoter. They were immunized intravenously with 5 micro g p33 and 48 hours later with 0.1 mg of an anti-CD40 antibody. All treated animals became diabetic, with mean time to onset 6 days but no animals that were injected with a control adenoviral protein rather than p33 did. This showed that anti-CD40 activated cytotoxic T lymphocytes for destruction of islet cells that expressed the 'transgenic self' antigen p33.

USE - The method is used, particularly with (I) as part of a vaccine, for treatment and prevention of infectious diseases, cancer and autoimmune diseases, e.g. prostatic or other cancers, leukemia, lymphoma, condyloma accuminatum, and infections by hepatitis, herpes simplex, cytomegalo or human immune deficiency viruses, parasites, bacteria or fungi.

ADVANTAGE - (II) provide effective priming of (I)-displaying APC, resulting in an efficient killer cell response and an improved protective effect.

Dwg.0/6

L16 ANSWER 4 OF 12 USPATFULL

ACCESSION NUMBER: 2000:9518 USPATFULL

TITLE: Activated dendritic cells and methods for their activation

INVENTOR(S): Maraskovsky, Eugene, Seattle, WA, United States

PATENT ASSIGNEE(S): Mc Kenna, Hilary J., Seattle, WA, United States
Immunex Corporation, Seattle, WA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 6017527		20000125
APPLICATION INFO.:	US 1996-763995		19961212 (8)

RELATED APPLN. INFO.: Continuation of Ser. No. US 1996-677762, filed on 10 Jul 1996, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Chan, Christina Y.
ASSISTANT EXAMINER: Gambel, Phillip
LEGAL REPRESENTATIVE: Henry, Janis C.
NUMBER OF CLAIMS: 15
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 3 Drawing Figure(s); 3 Drawing Page(s)
LINE COUNT: 1133
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB Antigen-expressing, activated dendritic cells are disclosed. Such dendritic cells are used to present tumor, viral or bacterial antigens to T cells, and can be useful in vaccination protocols. Other cytokines can be used in separate, sequential or simultaneous combination with the activated, antigen-pulsed dendritic cells. Also disclosed are methods for stimulating an immune response using the antigen-expressing, activated dendritic cells.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 5 OF 12 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2000309820 MEDLINE
DOCUMENT NUMBER: 20309820 PubMed ID: 10764746
TITLE: TTRAP, a novel protein that associates with CD40, tumor necrosis factor (TNF) receptor-75 and TNF receptor-associated factors (TRAFs), and that inhibits nuclear factor-kappa B activation.
AUTHOR: Pype S; Declercq W; Ibrahimi A; Michiels C; Van Rietschoten J G; Dewulf N; de Boer M; Vandenabeele P; Huylebroeck D; Remacle J E
CORPORATE SOURCE: Department of Cell Growth, Flanders Interuniversity Institute for Biotechnology, Campus Gasthuisberg, University of Leuven, Herestraat 49, B-3000 Leuven, Belgium.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (2000 Jun 16) 275 (24) 18586-93.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AJ251328; GENBANK-AJ269473
ENTRY MONTH: 200007
ENTRY DATE: Entered STN: 20000728
Last Updated on STN: 20020420
Entered Medline: 20000720

AB CD40 belongs to the **tumor necrosis factor** (TNF) receptor **family**. CD40 signaling involves the recruitment of TNF receptor-associated factors (TRAFs) to its cytoplasmic domain. We have identified a novel intracellular **CD40-binding protein** termed TRAF and TNF receptor-associated protein (TTRAP) that also interacts with TNF-R75 and CD30. The region of the CD40 cytoplasmic domain that is required for TTRAP association overlaps with the TRAF6 recognition motif. Association of TTRAP with CD40 increases profoundly in response to treatment of cells with CD40L. Interestingly, TTRAP also associates with TRAFs, with the highest affinity for TRAF6. In

transfected cells, TTRAP inhibits in a dose-dependent manner the transcriptional activation of a nuclear factor-kappaB (NF-kappaB)-dependent reporter mediated by CD40, TNF-R75 or Phorbol 12-myristate 13-acetate (PMA) and to a lesser extent by TRAF2, TRAF6, TNF-alpha, or interleukin-1beta (IL-1beta). TTRAP does not affect stimulation of NF-kappaB induced by overexpression of the NF-kappaB-inducing kinase (NIK), the IkappaB kinase alpha (IKKalpha), or the NF-kappaB subunit P65/RelA, suggesting it acts upstream of the latter proteins. Our results indicate that we have isolated a novel regulatory factor that is involved in signal transduction by distinct members of the TNF receptor **family**.

L16 ANSWER 6 OF 12 USPATFULL
ACCESSION NUMBER: 1999:141625 USPATFULL
TITLE: Isolated nucleic acid molecules useful as leukemia markers and in breast cancer prognosis and encoded polypeptides
INVENTOR(S): Rio, Marie-Christine, Illkirch, France
Tomasetto, Catherine, Strasbourg, France
Basset, Paul, Strasbourg, France
Byrne, Jennifer, Ashfield, Australia
PATENT ASSIGNEE(S): Bristol-Myers Squibb Company, Princeton, NJ, United States (U.S. corporation)
Institut National de la Sante et de la Recherche Medicale, Paris Cedex, France (non-U.S. corporation)
Centre National de la Recherche Scientifique, Paris Cedex, France (non-U.S. corporation)
Universite Louis Pasteur, Strasbourg Cedex, France (non-U.S. corporation)

	NUMBER	KIND	DATE	
PATENT INFORMATION:	US 5981218		19991109	
APPLICATION INFO.:	US 1996-691814		19960731	(8)

	NUMBER	DATE	
PRIORITY INFORMATION:	US 1995-2183P	19950809	(60)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Feisee, Lila
ASSISTANT EXAMINER: Kaufman, Claire M.
LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox P.L.L.C.
NUMBER OF CLAIMS: 48
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 53 Drawing Figure(s); 45 Drawing Page(s)
LINE COUNT: 7347
CAS INDEXING IS AVAILABLE FOR THIS PATENT.
AB The present invention relates to four novel human genes amplified and overexpressed in breast carcinoma and located on the q11-q21.3 region of chromosome 17. The four novel genes are useful in breast cancer prognosis. The present invention also relates to a fifth novel human gene expressed in breast carcinoma and located on chromosome 6q22-q23. A sixth novel gene is also described that is the murine homolog of the human D52 gene. The genes and gene fragments of the present invention are themselves useful as DNA and RNA probes for gene mapping by in situ hybridization with chromosomes and for detecting gene expression in human tissues (including breast and lymph node tissues) by Northern blot analysis.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 7 OF 12 WPIDS (C) 2002 THOMSON DERWENT
ACCESSION NUMBER: 2000-062029 [05] WPIDS
DOC. NO. NON-CPI: N2000-048592
DOC. NO. CPI: C2000-017141

TITLE: Novel proteins used to treat inflammatory diseases, NF-kappaB related diseases and for improvement of anti-tumor treatments.

DERWENT CLASS: B04 D16 S03

INVENTOR(S): HUYLEBROECK, D F E; PYPE, S M C; REMACLE, J E F J G

PATENT ASSIGNEE(S): (VLAA-N) VLAAMS INTERUNIVERSITAIR INST BIOTECHNOG

COUNTRY COUNT: 85

PATENT INFORMATION:

PATENT NO	KIND	DATE	WEEK	LA	PG
WO 9955859	A2	19991104	(200005)*	EN	48
RW: AT BE CH CY DE DK EA ES FI FR GB GH GM GR IE IT KE LS LU MC MW NL OA PT SD SE SL SZ UG ZW					
W: AL AM AT AU AZ BA BB BG BR BY CA CH CN CU CZ DE DK EE ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MD MG MK MN MW MX NO NZ PL PT RO RU SD SE SG SI SK SL TJ TM TR TT UA UG US UZ VN YU ZW					
AU 9939310	A	19991116	(200015)		
EP 1073739	A2	20010207	(200109)	EN	
R: AT BE CH CY DE DK ES FI FR GB GR IE IT LI LU MC NL PT SE					
JP 2002512796	W	20020508	(200234)		55
AU 752597	B	20020926	(200268)		

APPLICATION DETAILS:

PATENT NO	KIND	APPLICATION	DATE
WO 9955859	A2	WO 1999-EP3025	19990428
AU 9939310	A	AU 1999-39310	19990428
EP 1073739	A2	EP 1999-922165	19990428
		WO 1999-EP3025	19990428
JP 2002512796	W	WO 1999-EP3025	19990428
		JP 2000-546003	19990428
AU 752597	B	AU 1999-39310	19990428

FILING DETAILS:

PATENT NO	KIND		PATENT NO
AU 9939310	A	Based on	WO 9955859
EP 1073739	A2	Based on	WO 9955859
JP 2002512796	W	Based on	WO 9955859
AU 752597	B	Previous Publ.	AU 9939310
		Based on	WO 9955859

PRIORITY APPLN. INFO: EP 1998-201392 19980429

AN 2000-062029 [05] WPIDS

AB WO 9955859 A UPAB: 20000128

NOVELTY - An isolated functional protein (I) capable of interacting with the cytoplasmic domain of CD40 and/or other receptors of the **tumor necrosis factor** (TNF) receptor **superfamily** such as CD30 and TNF receptor I, where the protein has no homology to TNF receptor associated factor (TRAF)-proteins.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a nucleic acid sequence (II) encoding (I);

(2) a method for screening compounds comprising the use of (I);

(3) a compound isolated with the method of (2).

(4) a pharmaceutical composition comprising (I), or the compound of (3), and a pharmaceutically acceptable carrier; and

(5) use of (I) and/or a functional fragment for the manufacture of a pharmaceutical composition to treat TRAF, CD40, and/or NF-kappaB related diseases.

ACTIVITY - Antiarteriosclerotic; antiarthritic; neuroprotective; dermatological; immunosuppressive; antiinflammatory; immunosuppressive;

antiallergic.

MECHANISM OF ACTION - **CD40 binding proteins** which can be used as modulators of the CD40 signaling pathway.

USE - The proteins can be used to diagnose and treat TRAF-related, CD40-related, NF-kappaB related and/or Jun (kinase)-related diseases, and for the improvement of anti- tumor diseases (claimed). Diseases which may be treated include atherosclerosis (claimed), arthritis (claimed), multiple sclerosis (claimed), systemic lupus erythematosus (claimed), graft rejection (claimed), graft versus host disease, allergy, and autoimmune disease. The proteins can be used to sensitize tumor cells to antitumor treatments and to screen for compounds which interfere with the interaction of the proteins with other protein components of the TRAF, CD40 or NF-kappaB related pathway (claimed). The composition and antibodies can be used for detecting expression of CD40 related receptor associated proteins. The antibodies may also be used to purify the protein.

ADVANTAGE - None given.

Dwg.0/2

L16 ANSWER 8 OF 12 USPATFULL

ACCESSION NUMBER:	97:91164 USPATFULL
TITLE:	Method of preventing or treating disease characterized by neoplastic cells expressing CD40
INVENTOR(S):	Armitage, Richard J., Bainbridge Island, WA, United States Fanslow, III, William C., Federal Way, WA, United States Longo, Dan L., Kensington, MD, United States Murphy, William J., Frederick, MD, United States
PATENT ASSIGNEE(S):	Immunex Corporation, Seattle, WA, United States (U.S. corporation) The United States of America as represented by the Department of Health and Human Services, Washington, DC, United States (U.S. government)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5674492		19971007
APPLICATION INFO.:	US 1994-360923		19941221 (8)
RELATED APPLN. INFO.:	Continuation-in-part of Ser. No. US 1993-172664, filed on 23 Dec 1993, now abandoned		
DOCUMENT TYPE:	Utility		
FILE SEGMENT:	Granted		
PRIMARY EXAMINER:	Feisee, Lila		
ASSISTANT EXAMINER:	Gambel, Phillip		
LEGAL REPRESENTATIVE:	Perkins, Patricia Anne		
NUMBER OF CLAIMS:	18		
EXEMPLARY CLAIM:	1		
NUMBER OF DRAWINGS:	12 Drawing Figure(s); 6 Drawing Page(s)		
LINE COUNT:	1264		

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB There is disclosed a method of treating a mammal afflicted with a disease characterized by neoplastic cells that express CD40, comprising administering a therapeutically effective amount of a **CD40 binding protein** in a pharmaceutically acceptable buffer. **CD40 binding proteins** include monoclonal antibodies to CD40, and CD40 ligand. **CD40 binding proteins** may also be used to prevent disease characterized by neoplastic cells that express CD40, in individuals at risk for such disease.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L16 ANSWER 9 OF 12 MEDLINE DUPLICATE 2

ACCESSION NUMBER: 97189482 MEDLINE

DOCUMENT NUMBER: 97189482 PubMed ID: 9037712
TITLE: Construction and analysis of a detailed three-dimensional model of the ligand binding domain of the human B cell receptor CD40.
AUTHOR: Bajorath J; Aruffo A
CORPORATE SOURCE: Bristol-Myers Squibb Pharmaceutical Research Institute, Seattle, Washington 98121, USA.
SOURCE: PROTEINS, (1997 Jan) 27 (1) 59-70.
Journal code: 8700181. ISSN: 0887-3585.
PUB. COUNTRY: United States
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AB The interaction between the human B cell receptor CD40 and its ligand on T cells is critical for B cell proliferation and the regulation of humoral immune responses. CD40 is a member of the **tumor necrosis factor** receptor (TNFR) **family**. We report here the construction and analysis of a detailed three-dimensional model of the TNFR-homologous extracellular region of CD40. This study provides an example for structure-based model building in the presence of low sequence similarity. The assessment of model quality and sequence-structure compatibility is emphasized, and limitations of the model are discussed. The current CD40 model predicts structural details beyond the backbone level. Features of the CD40 ligand binding site are discussed in conjunction with the results of a previous mutagenesis study.

L16 ANSWER 10 OF 12 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 96029665 MEDLINE
DOCUMENT NUMBER: 96029665 PubMed ID: 7592751
TITLE: Presence of a new conserved domain in CART1, a novel member of the **tumor necrosis factor** receptor-associated protein **family**, which is expressed in breast carcinoma.
AUTHOR: Regnier C H; Tomasetto C; Moog-Lutz C; Chenard M P; Wendling C; Basset P; Rio M C
CORPORATE SOURCE: Institut de Genetique et de Biologie Moleculaire et Cellulaire, CNRS/INSERM/ULP, C.U. de Strasbourg, France.
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1995 Oct 27) 270 (43) 25715-21.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-X80200
ENTRY MONTH: 199512
ENTRY DATE: Entered STN: 19960124
Last Updated on STN: 19990129
Entered Medline: 19951214

AB CART1, a novel human gene, encodes a putative protein exhibiting three main structural domains: first, a cysteine-rich domain located at the amino-terminal part of the protein, which corresponds to an unusual RING finger motif; second, an original cysteine-rich domain located at the core of the protein and constituted by three repeats of an HC3HC3 consensus motif that we designated the CART motif, and which might interact with nucleic acid; third, the carboxyl-terminal part of the CART1 protein corresponds to a TRAF domain known to be involved in protein-protein interactions. Similar association of RING, CART, and TRAF domain was observed in the human **CD40-binding protein** and in the mouse **tumor necrosis factor** (TNF) receptor-associated factor 2 (TRAF2), both involved in signal transduction mediated by the TNF receptor **family** and in the developmentally

regulated Dictyostelium discoideum DG17 protein. CART1 is specifically expressed by epithelial cells in breast carcinomas and metastases. Moreover, in these malignant cells, the CART1 protein is localized in the nucleus. Altogether, these observations indicate that CART1 may be involved in TNF-related cytokine signal transduction in breast carcinoma.

L16 ANSWER 11 OF 12 MEDLINE
ACCESSION NUMBER: 95129692 MEDLINE
DOCUMENT NUMBER: 95129692 PubMed ID: 7530216
TITLE: A novel member of the TRAF family of putative signal transducing proteins binds to the cytosolic domain of CD40.
AUTHOR: Sato T; Irie S; Reed J C
CORPORATE SOURCE: La Jolla Cancer Research Foundation, Oncogene & Tumor Suppressor Gene Program, CA 92037.
SOURCE: FEBS LETTERS, (1995 Jan 23) 358 (2) 113-8.
Journal code: 0155157. ISSN: 0014-5793.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-L38509
ENTRY MONTH: 199502
ENTRY DATE: Entered STN: 19950307
Last Updated on STN: 19960129
Entered Medline: 19950223

AB CD40 is a member of the **tumor necrosis factor** receptor (TNF-R) **family** that regulates B-lymphocyte proliferation, immunoglobulin class-switching, and apoptosis through poorly defined signal transduction mechanisms. Using a yeast two-hybrid method, cDNAs were obtained that encode a novel protein, CD40-associated protein-1 (CAP-1), which binds specifically to the cytosolic domain of CD40 but not TNF-R1, TNF-R2, or Fas. The CAP-1 protein contains a C-terminal domain that shares strong amino acid sequence homology with a unique domain found recently in two putative signal transducing proteins that bind to the TNF-R2 cytosolic tail, TRAF1 and TRAF2. This C-terminal region of CAP-1 was sufficient to mediate binding to CD40 and homodimerization of CAP-1 proteins. The N-terminal portion of CAP-1 contains a RING finger motif and three zinc finger-like domains similar to those found in several regulatory proteins that interact with DNA or RNA. CAP-1 thus represents a new member of a **family** of potential signal transducing proteins that contain a conserved domain (the TRAF domain), bind to the cytosolic regions of particular members of TNF-R **family** proteins, and that can form homo- and heterotypic dimers.

L16 ANSWER 12 OF 12 MEDLINE DUPLICATE 4
ACCESSION NUMBER: 95073988 MEDLINE
DOCUMENT NUMBER: 95073988 PubMed ID: 7527023
TITLE: A novel RING finger protein interacts with the cytoplasmic domain of CD40.
AUTHOR: Hu H M; O'Rourke K; Boguski M S; Dixit V M
CORPORATE SOURCE: Department of Pathology, University of Michigan Medical School, Ann Arbor 48109.
CONTRACT NUMBER: CA61348 (NCI)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1994 Dec 2) 269 (48) 30069-72.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-U15637
ENTRY MONTH: 199412
ENTRY DATE: Entered STN: 19950116
Last Updated on STN: 19960129
Entered Medline: 19941230

AB CD40 is a member of the **tumor necrosis factor**

receptor **family** and, like other members, it appears to possess no intrinsic signaling capacity (e.g. kinase activity), suggesting that signal transduction is likely mediated by associating molecules. To identify such molecules, we have utilized the yeast two-hybrid system to clone cDNAs encoding proteins that bind the CD40 cytoplasmic domain. One such interacting protein, designated **CD40-binding protein**, has a N-terminal RING finger motif that is found in a number of DNA-binding proteins, including the V(D)J recombination activating gene RAG1. In addition, it contains a prominent central coiled-coil segment that may allow homo- or hetero-oligomerization. The C terminus possesses substantial homology to the **tumor necrosis factor** receptor-associated factor (TRAF) domain that is found in two proteins (TRAF1 and TRAF2) that associate with the cytoplasmic domain of the related 75-kDa **tumor necrosis factor** receptor. This is the first identification of a molecule that interacts with CD40 and whose sequence suggests a potential role in signaling.

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☐ 1: J Biol Chem 1999 May 14;274(20):14246-54

Related Articles, Links

CD40 signaling through tumor necrosis factor receptor-associated factors (TRAFs). Binding site specificity and activation of downstream pathways by distinct TRAFs.

Pullen SS, Dang TT, Crute JJ, Kehry MR.

Department of Biology, Boehringer Ingelheim Pharmaceuticals, Inc., Ridgefield, Connecticut 06877-0368, USA.

Tumor necrosis factor receptor-associated factors (TRAFs) associate with the CD40 cytoplasmic domain and initiate signaling after CD40 receptor multimerization by its ligand. We used saturating peptide-based mutational analyses of the TRAF1/TRAF2/TRAF3 and TRAF6 binding sequences in CD40 to finely map residues involved in CD40-TRAF interactions. The core binding site for TRAF1, TRAF2, and TRAF3 in CD40 could be minimally substituted. The TRAF6 binding site demonstrated more amino acid sequence flexibility and could be optimized. Point mutations that eliminated or enhanced binding of TRAFs to one or both sites were made in CD40 and tested in quantitative CD40-TRAF binding assays. Sequences flanking the core TRAF binding sites were found to modulate TRAF binding, and the two TRAF binding sites were not independent. Cloned stable transfectants of human embryonic kidney 293 cells that expressed wild type CD40 or individual CD40 mutations were used to demonstrate that both TRAF binding sites were required for optimal NF-kappaB and c-Jun N-terminal kinase activation. In contrast, p38 mitogen-activated protein kinase activation was primarily dependent upon TRAF6 binding. These studies suggest a role in CD40 signaling for competitive TRAF binding and imply that CD40 responses reflect an integration of signals from individual TRAFs.

PMID: 10318845 [PubMed - indexed for MEDLINE]

1: J Invest Dermatol 2000 Dec;115(6):1034-40 Related Articles, NEW Links

online

Distinct effects of CD30 and Fas signaling in cutaneous anaplastic lymphomas: a possible mechanism for disease progression.

Levi E, Wang Z, Petrogiannis-Haliotis T, Pfeifer WM, Kempf W, Drews R, Kadin ME.

Department of Pathology, Beth Israel-Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts 02215, USA.

Lymphomatoid papulosis is part of a spectrum of CD30+ cutaneous lymphoproliferative disorders characterized by spontaneous tumor regression. The mechanism(s) of regression is unknown. In a recent study, a selective increase in CD30 ligand expression in regressing lesions of lymphomatoid papulosis and cutaneous CD30+ anaplastic large cell lymphoma was shown, suggesting that activation of the CD30 signaling pathway may be responsible for tumor regression, whereas no difference in Fas/Fas ligand expression was found between regressing and nonregressing lesions. Therefore we tested the effects of CD30 and Fas activation on three CD30+ cutaneous lymphoma cell lines (Mac-1, Mac-2 A, JK) derived from nonregressing tumors of two patients who had progressed from lymphomatoid papulosis to systemic anaplastic large cell lymphoma. To evaluate the effects of CD30 signaling, the cell lines were incubated with a CD30 agonistic antibody, HeFi-1. Proliferative responses, mitogen-activated protein kinase, and nuclear factor kappa B activities were determined with and without CD30 activation. Mac-1 and Mac-2 A showed increased proliferative responses to incubation with CD30 activating antibody, HeFi-1. Inhibition of the mitogen-activated protein kinase activity caused growth inhibition of the Mac-1, Mac-2 A, and JK cell lines. Activation of the Fas pathway induced apoptosis in all three cell lines. Taken together, these findings suggest that resistance to CD30-mediated growth inhibition provides a possible mechanism for escape of cutaneous anaplastic large cell lymphoma from tumor regression. Mitogen-activated protein kinase inhibitors are potential therapeutic agents for the treatment of advanced cutaneous anaplastic large cell lymphoma. J Invest Dermatol 115:1034-1040, 2000

PMID: 11121138 [PubMed - indexed for MEDLINE]

1: Clin Chim Acta 2002 May 7;319(1):19-26

Related Articles, NEW Links

ELSEVIER SCIENCE
FULL-TEXT ARTICLE

Elevated soluble CD40 ligand is related to the endothelial adhesion molecules in patients with acute coronary syndrome.

Peng DQ, Zhao SP, Li YF, Li J, Zhou HN.

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BACKGROUND: Increasing evidence indicates that the CD40-CD40L interaction plays a pivotal role in the inflammatory regulation of atherosclerosis. Adhesion molecules especially the vascular adhesion molecules also play an important role in the pathogenesis of atherosclerosis which act as markers of inflammation. These inflammatory factors render vulnerability to the atherosclerotic plaque by triggering the fissure, rupture, and subsequent thrombosis, leading to the clinical scenario of unstable angina and acute myocardial infarction. METHODS: The difference of sCD40L concentration in different subtype of coronary heart disease and its relationship with vascular adhesion molecules was investigated. Enzyme-linked Immunosorbent Assay (EIA) was used to measure the serum sCD40L, soluble intercellular adhesion molecule-1 (sICAM-1), and soluble vascular cell adhesion molecule-1 (sVCAM-1). RESULTS: The sCD40L concentration was significantly higher in patients with acute coronary syndrome (ACS) (3.17+/-2.84 ng/ml) than in controls (1.19+/-1.05 ng/ml, $p<0.01$) and in patients with stable coronary heart disease (1.61+/-1.46 ng/ml, $p<0.05$). The sCD40L concentration was positively correlated with sICAM-1 ($r=0.413$, $p<0.01$), triglycerides (TG) ($r=0.23$, $p<0.05$), apoB ($r=0.248$, $p<0.05$), and HDL-cholesterol ($r=-0.253$, $p<0.05$). CONCLUSIONS: The sCD40L concentration was increased in acute coronary syndrome, suggesting the possible relation of CD40L to the pathogenesis. The serum CD40L concentration was positively correlated with adhesion molecule and was negatively associated with serum high-density lipoprotein cholesterol (HDL-C).

PMID: 11922919 [PubMed - indexed for MEDLINE]

1: N Engl J Med 1998 May 14;338(20):1413-21 Related Articles, NEW Links

Comment in:
- N Engl J Med. 1998 May 14;338(20):1461-3.

Epstein-Barr virus and a cellular signaling pathway in lymphomas from immunosuppressed patients.

Liebowitz D.

Marjorie B. Kovler Viral Oncology Laboratories, Department of Medicine, University of Chicago, IL 60637, USA.

BACKGROUND: Epstein-Barr virus (EBV) is associated with various malignant and benign lymphoproliferative disorders. It also efficiently transforms human B lymphocytes in vitro. The latent membrane protein 1 (LMP1) of EBV-infected cells plays a central part in this process by mimicking members of the family of tumor necrosis factor (TNF) receptors, thereby transmitting growth signals from the cell membrane to the nucleus through cytoplasmic TNF-receptor-associated factors (TRAFs). I sought evidence of LMP1-mediated signal transduction through TRAFs in tumor tissue from patients with post-transplantation lymphoproliferative disease and non-Hodgkin's lymphomas related to the acquired immunodeficiency syndrome (AIDS). METHODS: The association of LMP1 with TRAF-1 or TRAF-3 in tumor tissue was studied with double-immunofluorescence microscopy and immunoprecipitation assays. Evidence of LMP1-TRAF signaling was sought with an electrophoretic mobility shift assay for the nuclear factor-kappaB (NF-kappaB) transcription factor. RESULTS: Tumors from eight patients with post-transplantation lymphoproliferative disease, two patients with AIDS-associated non-Hodgkin's lymphoma, and three patients with endemic Burkitt's lymphoma were analyzed. Tumors from six of the patients with post-transplantation lymphoproliferative disease were positive for EBV and expressed LMP1; two samples were EBV-negative. Tumors from both patients with AIDS-associated non-Hodgkin's lymphoma were EBV-positive and expressed LMP1, whereas tumors from all three patients with Burkitt's tumors were positive for EBV but negative for LMP1. Double-immunofluorescence microscopy showed that LMP1 localized with and immunoprecipitated with TRAF-1 and TRAF-3 in all eight of the EBV-positive, LMP1-positive samples. An electrophoretic mobility shift assay revealed activated NF-kappaB in all eight EBV-positive, LMP1-positive samples as well, but not in either of the EBV-negative, LMP1-negative samples or in the three EBV-positive, LMP1-negative samples. CONCLUSIONS: LMP1-mediated signaling through the TRAF system has a role in the pathogenesis of the EBV-positive lymphomas that arise in immunosuppressed patients.

PMID: 9580648 [PubMed - indexed for MEDLINE]

1: J Immunol 2002 Feb 15;168(4):1600-9

Related Articles, Links

Prolonged blockade of CD40-CD40 ligand interactions by gene transfer of CD40Ig results in long-term heart allograft survival and donor-specific hyporesponsiveness, but does not prevent chronic rejection.

Guillot C, Guillonneau C, Mathieu P, Gerdes CA, Menoret S, Braudeau C, Tesson L, Renaudin K, Castro MG, Lowenstein PR, Anegon I.

Institut National de la Sante et de la Recherche Medicale, Institut de Transplantation et Recherche en Transplantation, Nantes, France.

Previous work on blockade of CD40-CD40 ligand interaction in mice and primates with anti-CD40 ligand mAbs has resulted in a moderate prolongation of allograft survival without the development of true allograft tolerance. In this study, we show in rats that adenovirus-mediated gene transfer of CD40Ig sequences into the graft resulted in prolonged (>200 days) expression of CD40Ig and in long-term (>300 days) survival. Recipients expressing CD40Ig displayed strongly (>90%) inhibited mixed leukocyte reactions and alloantibody production at early (days 5 and 17) and late time points (>100 day) after transplantation, but showed limited inhibition of leukocyte infiltration and cytokine production as evaluated by immunohistology at early time points (day 5). Recipients of long-surviving hearts showed donor-specific hyporesponsiveness since acceptance of second cardiac allografts was donor specific. Nevertheless, long-term allografts (>100 days) displayed signs of chronic rejection vasculopathy. Occluded vessels showed leukocyte infiltration, mainly composed of CD4(+) and CD8(+) cells, macrophages, and mast cells. These recipients also showed antidonor CTL activity. Recipients expressing CD40Ig did not show nonspecific immunosuppression, as they were able to mount anticognate immune responses that were partially inhibited at early time points and were normal thereafter. We conclude that gene transfer-mediated expression of CD40Ig resulted in a highly efficient inhibition of acute heart allograft rejection in rats. This treatment induced donor-specific inhibition of certain alloreactive mechanisms in the short-, but not the long-term, which resulted in long-term survival of allografts concomitant with the development of chronic rejection.

PMID: 11823487 [PubMed - indexed for MEDLINE]

Myeloma Treatment

Bisphosphonates

Chemotherapy

Peripheral Blood Stem Cell (PBSC) Transplantatio

Thalidomide

Emerging Treatments

Immunotherapy

Managing Fatigue

Myeloma Basics

MYELOMA DICTIONARY

Adhesion molecules: Complimentary molecules present on cell surfaces that allow cells to interact with each other, acting in the same way as a lock and key.

Allogeneic transplantation: Transplantation from a human donor who is not an identical genetic match.

Allogeneic Stem Cell Transplantation: A procedure in which bone marrow or peripheral blood stem cells from a donor (usually related) are collected, stored, and infused into a patient (recipient) following high-dose chemotherapy or radiation therapy.

Allograft: An allogeneic stem cell transplant.

Alpha interferon: Cytokine produced by T cells that exhibits a variety of immunomodulating effects, including suppression of cell growth, and enhancement of tumor cell killing.

Anemia: A decrease in the number of red blood cells in the blood.

Angiogenesis: The growth of new blood vessels.

Antibodies: Any of various proteins (immunoglobulins) that are generated in reaction to foreign proteins (antigens), thus producing an immunity against that protein.

Antigen: A substance that stimulates the production of an antibody to which it subsequently binds.

Antisense drug: Chemically altered stretch of DNA bases designed to bind to block the production of specific proteins.

Apheresis: A procedure in which blood is taken from a donor, a blood component (such as white blood cells, red blood cells, or plasma) is separated out, and the remaining blood components are reinfused back into the donor. With PBSC transplantation, the white blood cell component, which also contains the stem cells, is collected. In this case, the procedure may also be referred to as leukapheresis.

Apoptosis: Programmed (natural) cell death.

Autograft: An autologous stem cell transplant.

Autologous transplantation: Transplantation whereby the patient's own cells are reinfused.

Autologous Stem Cell Transplantation: A procedure in which a

patient's own stem cells from bone marrow or peripheral blood are collected, stored, and reinfused following high-dose chemotherapy or radiation therapy.

B cell: Also called a B lymphocyte. White blood cell that gives rise to a plasma cell after being exposed to a foreign substance.

Bence-Jones protein: See light chain.

Beta 2-microglobulin (β^2-microglobulin or β^2-M): A protein normally found on the surface of various cells in the body. Increased serum levels occur in inflammatory conditions and certain lymphocyte disorders, such as myeloma.

Biaxin: an antibiotic

Bisphosphonate: Type of drug used to treat osteoporosis and bone disease in cancer patients. Bisphosphonates work by inhibiting the activity of bone destroying cells called osteoclasts.

Blood urea nitrogen (BUN): A byproduct of protein metabolism that is normally filtered out of the blood and found in the urine. Elevated levels in the blood can indicate decreased kidney function.

Bone (skeletal) survey: A series of x-rays of the skull, spine, arms, ribs, and legs.

Bone marrow: Soft, spongy tissue found in the center of many bones where blood cells are produced.

Bone marrow aspiration: Removal of fluid and cells of the bone marrow via use of a needle.

Bone marrow biopsy: Removal of bone marrow tissue via the use of a needle.

Bone marrow microvessel density (MVD): A measure of the number of blood vessels in a bone marrow sample.

Bone marrow transplantation: A procedure in which stem cell-containing bone marrow is collected, stored, and infused following high-dose chemotherapy or radiation therapy.

C-reactive protein (CRP): A protein produced by the liver when there is an inflammatory process occurring in the body. Serum levels of CRP are increased in various inflammatory diseases, degenerative diseases, and cancers, including myeloma.

Calcium: Mineral important in bone formation. Elevated serum levels occur when there is bone destruction.

CD34+: A cell surface marker -CD stands for cluster of differentiation and the 34+ indicates a specific antigen for which this cell is positive. Stem cells are CD34+.

Chemotherapy: The use of drugs to treat cancer.

Chromosome: A thread-like structure in a living cell that contains genetic information.

Chromosome 13: Chromosomes are strands of DNA that are composed of genes containing instructions for all the production of body proteins. In some individuals with multiple myeloma, part of the short arm of chromosome 13 is deleted.

Chromosome analysis (cytogenetic testing): A laboratory test that measures the number and normalcy of chromosomes. Also known as cytogenetic testing.

Colony-Stimulating Factor (CSF): Protein that stimulates the development and growth of blood cells; sometimes called growth factor. Granulocyte colony-stimulating factor is a CSF that is used to mobilize stem cells from the bone marrow into the bloodstream prior to apheresis.

Complete Blood Count (CBC): Blood test that measures the number of red blood cells, white blood cells, and platelets in the blood and the relative proportions of the white blood cells present.

Computed tomography (CT): See computerized axial tomography (CAT).

Computerized axial tomography (CAT): Also known as computed tomography (CT). Imaging technique that uses a computer to generate 3-dimensional x-ray pictures.

Conventional chemotherapy: Chemotherapy that does not require stem cell rescue.

Corticosteroid: A potent class of drugs that has anti-inflammatory, immunosuppressive, and antitumor effects. Dexamethasone and prednisone are examples of corticosteroids.

Creatinine: A product of energy metabolism of muscle that is normally filtered out of the blood and found in the urine. Elevated levels in the blood can indicate decreased kidney function.

Cytokine: Soluble factor produced by cells that has an effect on other cells.

Cytogenetic testing: See chromosome analysis.

Decadron: also known as Dexamethasone, is a corticosteroid. See corticosteroids. It is part of the chemotherapy regimen called VAD.

Dendritic cell: Immune cell that plays an important role in initiating and regulating immune responses.

Dexamethasone: also known as Decadron, is a corticosteroid. See corticosteroids. It is used as part of the chemotherapy regimen called VAD.

DMSO: Dimethyl sulfoxide, a colorless chemical used for

cryopreservation of stem cells. When introduced into the body, may cause unpleasant or even serious toxic effects.

DNA (deoxyribonucleic acid): The genetic material of the cell located in the chromosomes.

Electrophoresis: Laboratory test used to measure the levels of various proteins in the blood or urine. Uses an electrical current to sort proteins by their molecular size.

Engraftment: The process in which stem cells in transplanted bone marrow or blood migrate to the bone marrow and begin to grow and produce new white blood cells, red blood cells, and platelets.

Erythropoietin: Growth factor that stimulates the bone marrow to produce red blood cells.

Farnesyl transferase: Enzyme involved in a signaling pathway that causes cancer cells to grow.

Filgrastim: A growth factor - GCSF (granulocyte colony stimulating factor) that stimulates the growth of white cells in the bone marrow.

Graft-versus-host disease (GVHD): Complication of allogeneic transplants resulting from donor immune cells recognizing the recipient's cells as foreign and mounting an attack against them.

Graft-versus-myeloma effect: Beneficial effect of allogeneic transplants resulting from the donor cells mounting an attack on the recipient's myeloma cells

Heavy chain: One of the long protein chains that make up an immunoglobulin molecule.

Hematologic: Pertaining to the blood.

Hematopoiesis: The formation and development of blood cells in the bone marrow.

Hemoglobin: The substance in the red blood cell that carries oxygen.

Hypercalcemia: Condition noted by elevated levels of calcium in the blood due to increased bone destruction.

Idiotype: Part of an antibody that determines exactly what antigen the antibody acts against.

Idiotype vaccine: A vaccine that uses the idiotype of an antibody as the antigen with which to stimulate an immune response.

Immune response: The interaction of an antigen with lymphocytes to induce the formation of antibodies.

Immune System: Network of related cells, tissues, and organs that protect the body from disease organisms, other foreign bodies, and cancers.

Immunoelectrophoresis (IEP): Also called immunofixation. Type of electrophoresis that uses a special antibody staining technique to identify specific types of immunoglobulin and light chains.

Immunofixation: See immunoelectrophoresis.

Immunoglobulin: An antibody that is produced by the plasma cell. Normally, it is made up of two types of proteins, one is called the heavy chain, the other the light chain.

Immunosuppressive drug: Drug given to suppress a patient's immune system, such as one given to prevent rejection of transplanted tissue.

Immunotherapy: The treatment of, or prevention against, a disease achieved through manipulation of the patient's immune system.

Institutional Review Board (IRB): A board designed to oversee the research process in order to protect participant safety. Made up of researchers, ethicists, and laypeople from the community, the board must review the trial protocols and the informed consent forms participants sign.

Interferon (IFN): A substance produced in the body by infected cells that protects noninfected cells from viral infection.

Interleukin -2 (IL-2):A cytokine (growth factor) that is produced by T-cells, which are lymphocytes.

Interleukin 6 (IL-6): A cytokine that promotes the growth and survival of myeloma cells.

Interleukin 12 (IL-2): Cytokine that promotes T cell function and tumor cell killing.

Lactate dehydrogenase (LDH): An enzyme found in body tissues. Elevated blood levels occur when there is tissue damage and may occur in myeloma, where they reflect tumor-cell burden.

Leukine: A granulocyte colony stimulating factor (G-CSF) that stimulates the growth of white blood cells in the bone marrow

Light chain: One of the short protein chains that make up an immunoglobulin molecule. May be of the kappa or lambda type. Light chains produced by myeloma cells are also referred to as Bence-Jones proteins.

Lymphocyte: Small white blood cell essential for normal function of the immune system; may be 1 of 2 types: a T lymphocyte or B lymphocyte.

Magnetic resonance imaging (MRI): Imaging technique that uses magnetic energy to provide detailed images of bone and soft tissue.

Maintenance therapy: Therapy used over a long period of time to prolong the length of remission.

Malignant: Cancerous, continuing to divide.

Matrix metalloproteinases (MMPs): Enzymes that break down the structure of connective tissue.

Melphalan: A chemotherapy agent (commercial name- Alkeran").

Mini-allograft: Type of allogeneic stem cell transplant that uses lower doses of chemotherapy or radiation and thus does not completely destroy the bone marrow; also known as mini-transplant or non-myeloablative transplant.

Mini-transplant: See Mini-allograft.

Monoclonal antibody: An identical copy of an antibody.

Monoclonal (M) protein: Identical immunoglobulin protein produced by myeloma cells. M protein is found in the blood or urine and is used as a marker for the amount of myeloma disease present in the body.

Monoclonal gammopathy of undetermined significance (MGUS): A precancerous and asymptomatic condition noted by the presence of M protein in the serum or urine. MGUS may progress to myeloma.

Morphology: Overall appearance.

Mucositis – Mouth and throat sores

Myeloablation: The killing of bone marrow by radiation or chemotherapy. This term usually refers to the complete or near-complete destruction of the bone marrow.

Nephrotoxicity: Toxicity to the kidneys.

Neutropenia: A below-normal number of neutrophils.

Neutrophil: A type of white blood cell that functions to destroy bacteria.

New Erythropoesis Stimulating Protein: a new protein that stimulates the bone marrow to produce red blood cells

Non-myeloablative transplant: See Mini-allograft.

Office for Human Research Protections (OHRP): This office safeguards participants in federally funded research and provides unity and leadership for many federal departments and agencies that carry out research involving human participants.

Osteoblast: Bone-forming cell.

Osteoclast: Bone-destroying cell that works in conjunction with bone-forming cells to repair bone.

Osteolytic lesion: Soft spot in the bone where bone tissue has been destroyed. The lesion appears as a hole on a standard bone x-ray.

Osteoporosis: Generalized bone loss typically associated with old age.

Palliative: Meant to reduce symptoms and relieve pain rather than to alter the course of disease.

Paraprotein: See monoclonal protein.

Peripheral Blood Stem Cell (PBSC): Stem cells collected from the blood. The term "peripheral" means that the cells come from outside the bone marrow.

Peripheral Blood Stem Cell (PBSC) Transplantation: A procedure in which blood containing mobilized stem cells is collected by apheresis, stored, and infused following high-dose chemotherapy or radiation therapy.

Placebo: A drug or treatment that is designed to look like the medicine being tested but that doesn't have the active ingredient. Placebos are rarely used in cancer treatment trials.

Plasma cell: Antibody-secreting immune cell that develops from a B cell.

Plasma Cell Labeling Index (PCLI): The percentage of plasma cells that are actively dividing.

Plasmablast: Immature plasma cell.

Plasmacytoma: Single tumor comprised of malignant plasma cells that occurs in bone or soft tissue. Patients with a plasmacytoma may develop myeloma.

Platelets: Small cell fragments in the blood that help it to clot.

Precursor cell: An earlier form of a cell, for example, B cells are precursors of plasma cells.

Protocol: An action plan for a clinical trial that includes detailed description of patients who may join the trial, the therapy that will be given, and the care the patients will receive during and after the trial.

Randomization: a method used to prevent bias in research; a computer or a table of random numbers generates treatment assignments, and participants have an equal chance to be assigned to one of tow or more groups (e.g., the control group or the investigational group)

Red blood cell: Oxygen-transporting blood cell.

Refractory disease: Disease that is not responsive to initial therapies or relapsed disease.

Relapse: Return of disease or disease progression.

Remission: The period during which no evidence of disease is present.

Salvage therapy: Second-line therapy; used to treat disease that has not responded to initial therapy or relapsed disease.

Samarium: a therapeutic radioisotope linked to a diphosphonate compound, which concentrates in bone.

Stem cell: Parent cell that grows and divides to produce red blood cells, white blood cells, and platelets. Found primarily in the bone marrow, but also in the peripheral blood.

Stem cell transplantation: Therapeutic procedure in which bone marrow or peripheral blood stem cells are collected, stored, and infused into a patient following high-dose chemotherapy to restore blood cell production.

Stromal cell: Structural cells of the bone marrow that help support and nourish the blood-producing cells.

Syngeneic Stem Cell Transplantation: A procedure in which bone marrow or peripheral blood stem cells from a patient's identical twin are collected, stored, and infused into the patient following high-dose chemotherapy or radiation therapy.

T cell: Also known as a T lymphocyte. Lymphocyte that plays an important role in immune responses and target cell killing.

T-lymphocytes: (also called T-cells) Cells of the immune system that play a key role in immune responses and targeted cell killing.

Tandem Transplant: Type of transplantation technique where a patient receives an autologous stem cell transplant followed by a mini-transplant two to four months afterward.

Vascular endothelial growth factor (VEGF): One of the major growth factors that promotes angiogenesis.

White blood cell: Also called a leukocyte. One of the major cell types in the blood. Responsible for immune defenses.

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